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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 08:32:27 ON 28 AUG 2000

=> file medline biosis embase caplus uspatfull

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ENTRY

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0.21

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FILE 'MEDLINE' ENTERED AT 08:32:39 ON 28 AUG 2000

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FILE 'CAPLUS' ENTERED AT 08:32:39 ON 28 AUG 2000

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FILE 'USPATFULL' ENTERED AT 08:32:39 ON 28 AUG 2000

CA INDEXING COPYRIGHT (C) 2000 AMERICAN CHEMICAL SOCIETY (ACS)

=> s cytokine (s) igg2 (s) fusion

L1 0 CYTOKINE (S) IGG2 (S) FUSION

=> s cytokine (s) igg4 (s) fusion

L2 5 CYTOKINE (S) IGG4 (S) FUSION

=> dup rem l2

PROCESSING COMPLETED FOR L2

L3 3 DUP REM L2 (2 DUPLICATES REMOVED)

=> d l3 ibib kwic

L3 ANSWER 1 OF 3 MEDLINE

DUPLICATE 1

ACCESSION NUMBER: 1999247563 MEDLINE

DOCUMENT NUMBER: 99247563

TITLE: Improving the efficacy of antibody-interleukin 2 fusion proteins by reducing their interaction with Fc receptors.

AUTHOR: Gillies S D; Lan Y; Lo K M; Super M; Wesolowski J

CORPORATE SOURCE: Lexigen Pharmaceuticals Corporation, Lexington, Massachusetts 02421-3125, USA.. sgillies@lexigenpharm.com

SOURCE: CANCER RESEARCH, (1999 May 1) 59 (9) 2159-66.

Journal code: CNF. ISSN: 0008-5472.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals; Cancer Journals

ENTRY MONTH: 199907

ENTRY WEEK: 19990704

AB **Fusion** proteins between whole antibodies (Abs) and **cytokines** (immunocytokines) such as interleukin 2 have shown

efficacy in several mouse tumor models despite a circulating half-life that is significantly. . . isotype of the human heavy chain C region from IgG1 or IgG3 to those with reduced binding to FcR, e.g., **IgG4**. The same effect could also be achieved through site-directed

mutagenesis

of the FcR binding site in the IgG1 H chain.. . . cells showed increased binding of interleukin 2-based immunocytokines, relative to their corresponding Abs, and that this was reversed in those **fusion** proteins made with **IgG4** or mutated IgG1 H chains. All of the **fusion** proteins showing reduced FcR binding also had reduced Ab-dependent cellular cytotoxicity activity, as measured in 4-h chromium release assays. A complete loss of complement-dependent cytotoxicity activity was seen with an **IgG4**-based immunocytokine derived from an IgG1 Ab with potent activity. Despite these reduced effector functions, the **IgG4**-based immunocytokines with extended circulating half-lives showed equivalent (in the case of severe combined immunodeficiency mouse xenograft models) or better (in. . .

=> d 13 ibib kwic total

L3 ANSWER 1 OF 3 MEDLINE DUPLICATE 1
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DOCUMENT NUMBER: 99247563
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AUTHOR: Gillies S D; Lan Y; Lo K M; Super M; Wesolowski J
CORPORATE SOURCE: Lexigen Pharmaceuticals Corporation, Lexington, Massachusetts 02421-3125, USA.. sgillies@lexigenpharm.com
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L3 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:608639 CAPLUS
DOCUMENT NUMBER: 129:229689
TITLE: Chimeric polypeptides containing chemokine domains
INVENTOR(S): Herrmann, Stephen H.; Swanberg, Stephen L.
PATENT ASSIGNEE(S): Genetics Institute, Inc., USA
SOURCE: PCT Int. Appl., 69 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9838212	A2	19980903	WO 1998-US4002	19980227
WO 9838212	A3	19990114		
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG			
US 6100387	A	20000808	US 1997-808720	19970228
AU 9864440	A1	19980918	AU 1998-64440	19980227
EP 1012309	A2	20000628	EP 1998-910117	19980227
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI			
WO 9920759	A1	19990429	WO 1998-US22282	19981021
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
AU 9911105	A1	19990510	AU 1999-11105	19981021
EP 1025229	A1	20000809	EP 1998-953836	19981021
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI			
PRIORITY APPLN. INFO.:			US 1997-808720	19970228
			US 1997-955826	19971022
			WO 1998-US4002	19980227
			US 1998-175713	19981020
			WO 1998-US22282	19981021

IT **IgG4**

Immunoglobulin fusion products

RL: BAC (Biological activity or effector, except adverse); BPN (Biosynthetic preparation); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(**fusion** products with **cytokines**; construction and
biol. activity of chimeric polypeptides contg. chemokine domains)

L3 ANSWER 3 OF 3 USPATFULL

ACCESSION NUMBER: 97:6049 USPATFULL
TITLE: Method of refolding human IL-13
INVENTOR(S): Culpepper, Janice, Mountain View, CA, United States
McKenzie, Andrew, Redwood City, CA, United States
Dang, Warren, San Jose, CA, United States
Zurawski, Gerard, Redwood City, CA, United States
PATENT ASSIGNEE(S): Schering Corporation, Kenilworth, NJ, United States
(U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5596072	19970121
APPLICATION INFO.:	US 1993-12543	19930201 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1992-933416, filed on 21 Aug 1992, now abandoned	
DOCUMENT TYPE:	Utility	
PRIMARY EXAMINER:	Draper, Garnette D.	

ASSISTANT EXAMINER: Spector, Lorraine M.
LEGAL REPRESENTATIVE: Ching, Edwin P.
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 288 Drawing Figure(s); 61 Drawing Page(s)
LINE COUNT: 4619

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD . . . purified naive surface IgD+ human B cells in the presence of
IL-4 or IL-13 (Table 7). Considerable levels of IgM, **IgG4**,
total IgG and IgE, but no IgA were produced. There was no IgA
production

is compatible with previous observations which. . . Ig production.
Inhibition of total IgG production by CD40Ig could not be measured,
since the Ig portion of the CD40-Ig **fusion** protein gave a
strong signal in the IgG ELISA. Interestingly, Ig production, including
IgG4 and IgE production, induced by IL-13 in the presence of
COS-7/CD40L cells was not blocked by anti-IL-4 mAbs (10 .mu.g/ml),. . .
. 7). These results indicate that IL-13 induces Ig production
independently from IL-4. These data furthermore indicate that IL-13 is
another **cytokine** that directs naive surface IgD+ human B cells
to switch to **IgG4** and IgE producing cells in the presence of a
contact-mediated costimulatory signal delivered by COS-7 cells
expressing the mouse or. . .

=> d his

(FILE 'HOME' ENTERED AT 08:32:27 ON 28 AUG 2000)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS, USPATFULL' ENTERED AT 08:32:39 ON
28 AUG 2000

L1 0 S CYTOKINE (S) IGG2 (S) FUSION
L2 5 S CYTOKINE (S) IGG4 (S) FUSION
L3 3 DUP REM L2 (2 DUPLICATES REMOVED)

=> s igg2 (s) fusion (s) protein

L4 107 IGG2 (S) FUSION (S) PROTEIN

=> dup rem

ENTER L# LIST OR (END):14

PROCESSING COMPLETED FOR L4

L5 67 DUP REM L4 (40 DUPLICATES REMOVED)

=> s igg2 (s) fusion (s) protein (s) lymphokine

L6 0 IGG2 (S) FUSION (S) PROTEIN (S) LYMPHOKINE

=> s igg2 (s) fusion (s) protein (p) lymphokine

L7 0 IGG2 (S) FUSION (S) PROTEIN (P) LYMPHOKINE

=> s igg2 (s) fusion (s) protein (p) chemokine

L8 0 IGG2 (S) FUSION (S) PROTEIN (P) CHEMOKINE

=> s igg2 (s) fusion (s) protein (p) interleukin

L9 3 IGG2 (S) FUSION (S) PROTEIN (P) INTERLEUKIN

=> dup rem 19

PROCESSING COMPLETED FOR L9

=> d l10 ibib kwic

L10 ANSWER 1 OF 1 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000107064 MEDLINE
DOCUMENT NUMBER: 20107064
TITLE: A novel Leishmania infantum recombinant antigen which
elicits interleukin 10 production by peripheral blood
mononuclear cells of patients with visceral
leishmaniasis.
AUTHOR: Suffia I; Ferrua B; Stien X; Mograbi B; Marty P; Rousseau
D; Fragaki K; Kubar J
CORPORATE SOURCE: Groupe de Recherche en Immunopathologie de la
Leishmaniose,
Laboratoire de Parasitologie, Faculte de Medecine, 06107
Nice Cedex 2, France.
SOURCE: INFECTION AND IMMUNITY, (2000 Feb) 68 (2) 630-6.
Journal code: GO7. ISSN: 0019-9567.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 200004
ENTRY WEEK: 20000402
AB We report here the characterization of a novel Leishmania infantum
protein termed papLe22 (22-kDa potentially aggravating
protein of Leishmania). A positive clone from a cDNA library was
identified by serum of a visceral leishmaniasis (VL) patient. Full-length
cDNA obtained using rapid amplification of cDNA ends-PCR codes for a
22-kDa **protein**. In L. infantum promastigotes an endogenous
nuclear **protein** of 14-kDa electrophoretic mobility was found by
using an antiserum prepared against the **fusion protein**
glutathione S-transferase-papLe22. Its expression was also shown in L.
infantum amastigotes and in Leishmania major and Leishmania guyanensis
promastigotes. VL patients' sera showed anti-papLe22 immunoglobulin M
(IgM) and IgG reactivities, indicating that a primary response against
the
leishmanial **protein** papLe22 accompanied acute VL manifestations.
Specific IgG levels were correlated with patients' clinical status. The
presence of IgG1, **IgG2**, and IgG3 subclasses suggested a mixed
Th1- and Th2-type response; there was no correlation between subclass
reactivity and the disease course. The recombinant papLe22 specifically
activated **interleukin**-10 production by VL patients' peripheral
blood mononuclear cells collected at diagnosis and after
treatment-induced
cure, indicating its contribution to VL. . .

=> log y

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	46.43	46.64

STN INTERNATIONAL LOGOFF AT 08:39:01 ON 28 AUG 2000